MATTERS ARISING

Mycoplasma genitalium: a cause of non-gonococcal urethritis?

In a recent issue of this journal1 Horner and Taylor-Robinson expressed concern about the criteria applied for selection of the control group in a study on the prevalence of Mycoplasma genitalium reported by us.2 We found a significant association between the presence of M genitalium and symptoms and signs of non-gonococcal urethritis (NGU), whereas no association was found between Ureaplasma urealyticum and NGU.

M genitalium is an extremely slow-growing and fastidious microorganism isolated more than 10 years ago.3 Although considerable time and efforts have been spent to gain more knowledge of its significance, reliable studies had to await the development of sensitive non-culture methods such as the polymerase chain reaction (PCR).4

The main objection expressed by Horner and Taylor-Robinson concerns the selection of the control group. At enrolment all of our patients were examined clinically and only patients without any discharge and without complaints of urethritis were included in the control group. In Danish STD clinics it is common practice to do microscopy of urethral smears only if the patients have complaints of urethritis; however, all patients have swabs taken for culture of Neisseria gonorrhoeae and Chlamydia trachomatis. Thus, we selected the control group among patients who would not have had an urethral specimen taken had they not visited an STD clinic. In our opinion the two groups studied represent closely the everyday clinical situation.

Having said that, we admit that the authors raise an important question. "Asymptomatic patients" in the control group would consequently be patients without complaints and with no discharge but with $\geqslant 5$ polymorphonuclear leucocytes/high-power microscopic field. We do not feel that these patients should be included in the study group, but it might be reasonable to consider them as a separate group. We cannot exclude the possibility that exclusion of "asymptomatic NGU" patients from the control group might have changed the pattern of possible pathogens found in that group. If some or even all of the M genitalium positive patients in the control group had "asymptomatic NGU" as implied by Horner and Taylor-Robinson,1 the association would have been strengthened. Obviously, excluding patients with "microscopic urethritis" from the control group might have decreased the percentage of ureaplasma positive patients in this group, but we find it hard to believe that the conclusions regarding the association of U urealyticum with NGU would have changed; after all we found 33% ureaplasma positives in the NGU group as compared with 47% in the control group.

What we believe to be more important for estimates of the true prevalence of M genitalium is the fact that all previously published studies based on PCR have used

primers selected from the main adhesin gene MgPa. From results recently obtained by amplification of overlapping fragments of the MgPa gene from four new urethral isolates obtained by culture of specimens from patients enrolled in the above mentioned study we found that this gene shows considerable genetic variation. Out of 10 different primer-combinations, only five correctly detected vast amounts of *M genitalium* DNA from all four Danish strains. Therefore, the prevalence of M genitalium found in previous studies might represent only the minimum figure, since strains with MgPa gene sequences different from the type-strain would not be detected.

This risk of a low detection rate is even more pronounced when nested PCR is applied as was the case in the study published by Horner et al.5

It is obvious that we do not know enough to establish a role of M genitalium as a urogenital pathogen. Criticism could be raised against the selection of patients enrolled in our study² and possible technical insufficiencies in the study by Horner et al.5 It is encouraging, though, that the two studies obtain similar results, namely that M genitalium DNA could be detected in urethral specimens from 27% versus 23% of patients with NGU and in 9% versus 6% of the control patients in our study² and the study by Horner et al,5 respectively.

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Infectious osteitis pubis in an HIV seropositive female

Desmond and colleagues1 report a case of infectious osteitis pubis arising in a HIV seropositive woman following a termination of pregnancy. They have no firm details of the surgical procedure but reasonably assume that it was a vacuum termination and know that it was performed under local anaesthesia. They comment that uterine perforation is more common where the uterus is retroverted (as was the case in their patient) and suggest that the termination "may have been complicated by an anterior wall perforation resulting in contiguous infection of the pubic symphysis. . .'

As a practising gynaecologist I would confirm that perforation in these circumstances is a rare but recognised complication. It is unlikely to have occurred unrecognised in a patient under local anaesthesia and-of more relevance-an anterior

perforation of the uterus implies entry into the bladder or more commonly the peritoneal cavity and not the symphysis which would lie inferiorly to any potential site of perforation. If the association between termination of pregnancy and pubic osteitis in their patient is anything more than coincidence, it is most likely to be due to haematogenous spread of infection, rather than from direct surgical trauma.

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Desmond N, Bignardi GE, Coker RJ, Grech P, Harris JRW. Infectious osteitis pubis in an HIV seropositive female. *Genitourin Med* 1994;70:127-9.

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